discrete and in one instance confluent miliary nodules in the parenchyma, lesions that can be correctly estimated as of recent development, while, on the other hand, the lesions present in Case III (Figs. 2 and 3) are evidently very old, as evidenced by the extensive coagulation necrosis and the reactive fibrotic condition in the immediate neighborhood.

SUMMARY. We have given clinical and pathological proof of the occurrence of cases of ascending renal tuberculosis, ascending at least in the sense that the renal and ureteral lesions are secondary to the bladder involvement; we have shown that minimal tuberculous renal lesions, when associated with extensive vesical and ureteral changes, are doubtless, in some cases, later involvements of the urinary tract, be they produced in the true ascending, canalicular sense of propagation by contiguity or in a more circuitous fashion by late embolic invasion of the kidney; and, by the results of the removal of the kidney in two of the cases, have given ample testimony of the value of nephrectomy even in this type of urinary tuberculosis. Although the renal parenchyma is practically uninvolved in some of these cases the retention of tuberculous urine in the pelvis of the kidney and the constant contamination of the bladder with tuberculous products elaborated in the ureter are sufficiently active factors in interfering with recovery.

EFFECTS OF THE INJECTION OF ATROPIN ON THE PULSE-RATE, BLOOD-PRESSURE AND BASAL METABOLISM IN CASES OF "EFFORT SYNDROME."

BY CYRUS C. STURGIS, FIRST LIEUT., M.C., U.S.A.,
PENDLETON, OREGON,

JOSEPH T. WEARN, M.D., FIRST LIEUT., M.C., U.S.A.,
CHARLOTTE, N. C.,

AND

EDNA H. TOMPKINS,
CAMBRIDGE, MASS.

(From the Cardiovascular Division of the Medical Service, U. S. Army General Hospital No. 9, Lakewood, New Jersey.)

INTRODUCTION. During the studies upon the "Irritable Heart of Soldiers" at U. S. Army General Hospital No. 9, it was found that a considerable percentage of the cases were responding in a characteristic way to standard injections of epinephrin,[1] while others of the group reacted no more to the drug than did a normal individual. In patients sensitive to the drug there was in each instance a rather marked increase in the pulse-rate. This effect on the heart-rate is probably due to the action of epinephrin on the accelerator fibers to the heart. In view of this fact, it was decided to study further the nervous control of the heart in soldiers with "effort syndrome," by observing the action of atropin sulphate on the vagus control.

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[1] Wearn, J. T., and Sturgis, C. C.: Effects of the Injection of Epinephrin in Soldiers with "Irritable Heart." (To be published.)

The patients studied gave a history of the symptoms and signs of "effort syndrome" or "irritable heart," dating back in some of them to childhood, and in all of them a number of years. Some of them were epinephrin sensitive and others were not.

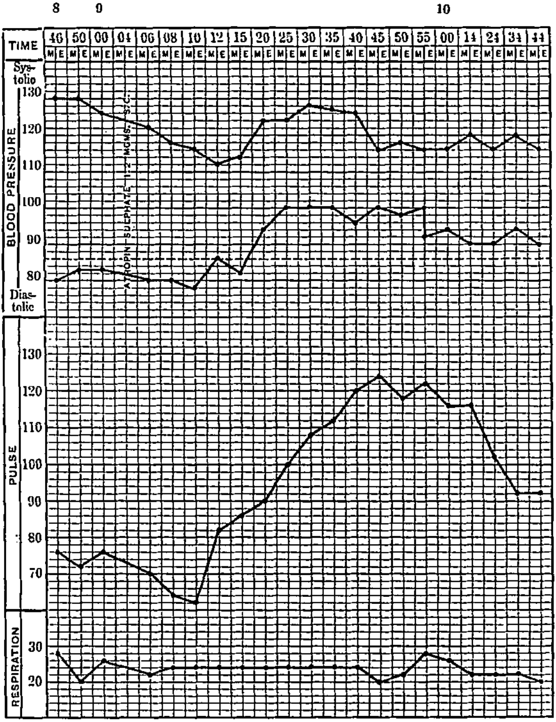

Chart showing the method of recording results after subcutaneous injection of atropin.

METHOD. The patient was rested in bed for one hour or longer until the pulse-rate and blood-pressure readings were constant.

in 9 of the 10 subjects. This drop was followed immediately by a rise in the pulse-rate varying from six to thirty beats per minute.

In 9 of the 10 normal subjects there was an increase in the diastolic blood-pressure, and 8 showed a drop in systolic blood-pressure. All of the normal men showed a drop in pulse-pressure. This drop in pulse-pressure occurred before the maximum increase in pulse-rate in 6; in 2 others it followed the maximum rise, and in the remaining 2 it occurred simultaneously with the maximum increase in pulse-rate.

TABLE II.—SHOWING PULSE-RATE CHANGES AND THEIR RELATIONS TO INJECTIONS OF ATROPIN SULPHATE IN SOLDIERS WITH "IRRITABLE HEART" OR "EFFORT SYNDROME" AND IN NORMAL MEN.

	Number of subjects.	Number showing drop in pulse-rate.	Average maximum drop in pulse-rate.	Number showing increase in pulse-rate following the drop.	Average time of beginning increase in pulse-rate after injection.	Average maximum pulse-rate increase.	Average time of maximum pulse-rate increase after the injection.
Cases of Effort syndrome	21	19	10 beats per min.	20	8 min.	30.4 beats per min.	37.4 min.
Normal men .	10	9	10.2 beats per min.	9	10.5 min.	21.8 beats per min.	39 min.

TABLE III.—SHOWING CHANGES IN BLOOD-PRESSURE FOLLOWING INJECTION OF ATROPIN SULPHATE INTO SOLDIERS WITH "IRRITABLE HEART" OR "EFFORT SYNDROME" AND INTO NORMAL MEN.

	Number of subjects.	Number showing a rise in diastolic blood-pressure.	Average rise in diastolic blood-pressure.	Number showing drop in systolic blood-pressure.	Number showing rise in systolic pressure.	Number showing drop in pulse-pressure.	Average drop in pulse-pressure.
Cases of Effort syndrome	21	20	11.9 mm. mercury	11	10	14	14.5 mm. mercury
Normal men .	10	9	7.8 mm. mercury	8	0[1]	10	10.8 mm. mercury

There was little difference in the response of the "effort syndrome" cases to the drug. As is shown in Tables II and III the most noticeable differences are the greater increase in pulse-rate in the cases with "effort syndrome," and the fact that on the average the increase in rate began sooner than it did in the normal controls.

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[1] Two showed no change in systolic blood-pressure.

The changes in blood-pressure were about the same in both groups. One rather striking change which was common to both groups was the fact that the compensatory drop in pulse-pressure was caused more by a rise in the diastolic than by a fall in the systolic blood-pressure. This rise in diastolic pressure varied from 4 to 25 mm. of mercury.

In the "irritable heart" cases and in the normal controls, dilatation of the pupils was noted, and dryness of the mouth was frequently complained of.

METABOLISM RESULTS. Higgins and Means[3] reviewed the literature upon the respiratory exchange under atropin and added the reports of studies upon themselves as normal subjects. They concluded there was a slight elevation in metabolism and an increased dead space due to dilatation of the bronchi after therapeutic doses of atropin.

The metabolism was studied by us in 6 cases under the effects of atropin. The time interval between the injection and the metabolism determination depended upon the pulse curve after the administration of the drug. In 4 of the cases a period was taken shortly after the injection, at the time of the pulse depression which ordinarily precedes the later rise. Two of these showed a small drop in the heat production and two showed rises so slight as to be within the upper limit of error. They gave no noteworthy change in respiratory rate, volume or quotient.

In each case one period was run as nearly as possible at the time of the greatest pulse acceleration. Under that condition three of the patients showed a small drop in the metabolism and three no change worthy of mention. This is interesting in view of the often discussed problem of how great a part increased heart activity plays in the elevation of metabolism under various conditions, and may be of some value as partial evidence that the increase in metabolism that occurs after epinephrin injection is not due to acceleration of the pulse. The respiratory quotient dropped slightly in each case. The respiratory rate and volume did not vary beyond a normal basal limit. Table IV summarizes these results.

SUMMARY. In cases of "irritable heart" and in normal men after injection of atropin sulphate there was generally a preliminary drop followed by an increase in pulse-rate. This increase was greater on the average in the "irritable heart" cases than in the normal controls. There was also a drop in pulse-pressure in the majority of the subjects, dependent upon a rise in diastolic blood-

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[3] Jour. Pharmacol. and Exper. Therap., 1915, vii, 1.

TABLE IV.

Case No.	Name.	Age.	Height.	Weight.	Period No.	Minutes from injection to period start.	CO_2 per min.	O_2 per min.	R. Q.	Pulse.	Resp.	Vol. per min. S.T.P.D.	Vol. per resp.	Cal. per sq. meter per hour.	Metabolism, per cent. from normal.	Amount of metabolic change.
			Cm.	Kg.			C.c.	C.c.					C.c.			
1	G. A.	24	174	68.4	1–2	..	212	251	0.85	75	14.6	5.76	396	39.9	+ 1	
					3	5	198	241	0.82	59	14.3	5.29	371	38.1	− 4	− 5
					4	37	203	246	0.83	99	14.0	5.51	394	39.0	− 1	− 2
2	D. C.	26	177	64.1	1	..	211	260	0.81	59	13.3	5.45	411	41.7	+ 6	
					2	3	211	269	0.78	59	15.5	5.54	358	42.9	+ 9	+ 3
					3	45	214	273	0.78	92	15.7	5.61	357	43.4	+10	+ 4
3	R. D.	23	173	51.6	1	..	185	209	0.89	70	12.4	4.92	398	38.2	+ 3	
					2	17	176	210	0.84	102	12.3	4.74	387	38.0	+ 4	+ 1
4	E. G.	24	165	58.9	1–2	..	194	238	0.82	70	11.4	5.57	491	41.7	+ 6	
					3	3	201	247	0.81	64	11.2	5.67	508	43.3	+10	+ 4
					4	28	171	225	0.76	91	11.5	5.02	436	38.8	− 2	− 8
5	M. L.	19	167	62.0	1	..	195	220	0.85	76	16.1	5.72	355	38.8	− 2	
					2	46	170	217	0.78	97	13.8	5.02	363	36.2	− 8	− 6
6	H. R.	24	169	60.2	1–2	..	201	264	0.77	69	31.2	7.31	236	44.8	+13	
					3	3	190	248	0.77	64	40.8	8.57	210	42.2	+ 7	− 6
					4	42	189	251	0.75	103	31.5	7.97	253	42.4	+ 7	− 6

pressure in the majority of cases and upon an additional drop in systolic blood-pressure in some of the cases.

There was no increase in basal metabolism after the subcutaneous injection of atropin sulphate.

There was a slight fall in the basal metabolism in three of the six cases studied.

There was always a slight drop in the respiratory quotient.

POINTS OF DIFFERENTAL DIAGNOSTIC VALUE IN PULMONARY ABSCESS, BRONCHIECTASIS AND PULMONARY TUBERCULOSIS, WITH AN EXPLANATION OF THE CAUSE OF THE DIFFERENCE IN AUSCULTATORY FINDINGS.[1]

By Francis Marion Pottenger, A.M., M.D., LL.D.,
MONROVIA, CALIFORNIA.

Pulmonary abscess, bronchiectasis and pulmonary tuberculosis are very often wrongly diagnosed. Unless one has had considerable opportunity to examine patients suffering from each of these diseases, he is liable to experience considerable difficulty in differentiating them. If, however, a careful study of the disease and the manner in which it affects the patients is made the differentiation will not be so difficult. Both pulmonary abscess and bronchiectasis are often diagnosed as tuberculosis on the basis of cough and expectoration. The fact that bacilli are not found is no longer sufficient to prove the non-tuberculous nature of a pulmonary process; consequently, this fact confuses more than it helps. In my own work I have had opportunity to study numbers of patients who were expectorating large quantities of sputum. Some of these have been of a tuberculous nature, yet bacilli have been found only at rare intervals. In other patients who had had a definite tuberculous process which had been accompanied by cavity formation and sputum containing bacilli the general process healed, leaving a secreting cavity, with a considerable amount of expectoration in which tubercle bacilli were not found, even after repeated examination. So while the presence of tubercle bacilli shows the definite tubercular nature of sputum, their absence does not prove the process to be non-tuberculous.

Clinical History. Pulmonary abscess usually follows acute infection of the lung, in which such organisms as streptococci, staphylococci or the pneumococci are the etiological factors, such as have been common during the epidemics in both army and civil life during the past two years. It also follows operations on the

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[1] Read before the Annual Meeting of the American Climatological and Clinical Association, at Atlantic City, June 14, 16 and 17, 1919.